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THE

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VOL. I.—11TH YEAR. SYDNEY: SATURDAY, JUNE 28, 1924. No. 26.

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Table of Contents

CLINICAL LECTURE ON SYPHILIS IN RELATION TO GASTRIC DISORDERS.[1]

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SYPHILIS of the stomach was formerly considered so rare an event that its very existence was questioned. Many medical practitioners today do not think of it as a possible factor in gastric disorders which present themselves for treatment. I remember that the first time its possible existence was seriously brought to my attention was in the illuminating address on "Abdominal Syphilis" delivered by Mr. Fred Bird as the Presidential Address in the Section of Surgery at the Australasian Medical Congress in Adelaide in 1905. That address still repays study and if you turn it up, you will find two cases of gastric syphilis described in full.

Of recent years much more attention has been paid to its existence. It has been said by Ensterman that our knowledge of gastric syphilis may be divided into three periods: The first from 1891 to 1905 in which evidence of the condition was obtained chiefly from *post mortem* material; the second from 1905 to 1910 when knowledge was increased by cases being reported in which the diagnosis rested on clinical history and the response to anti-syphilitic treatment; and the third from 1910 to the present day in which serological and radiological examination have so greatly aided in diagnosis.

Apart from temporary gastric manifestations in the secondary stage of syphilis, gastric syphilis is usually described as a tertiary manifestation. And as in syphilis of the lung, it is said to show itself in a general mutilating sclerosis; or less frequently as a gumma or gummatous ulcer. But when we seek to form any clear idea as to its usual pathological or clinical manifestations, we are beset with descriptions of such multitudinous forms and so variable an array of symptoms and signs that we run the danger of imagining it to be present in all sorts of gastric conditions. When in this danger, it is well to remind ourselves that though gastric syphilis is more common than we used to suppose, it still remains true that the intestinal tract is the least commonly affected of all the great visceral systems and that of all the intestinal tract the stomach is the least often affected with syphilis.

There are thus the two dangers awaiting you in practice: the first, that for want of thought you may fail to recognize an apparent carcinoma as really a curable condition, the second that by over

[1] Delivered in the Medical School of the University of Adelaide.

emphasis on the undoubted occurrence of syphilis of the stomach, you may ascribe to that infection conditions which are really due to other causes.

The open mind, prepared to observe carefully, to use every known method of investigation and to weigh evidence carefully, is just as necessary here as in all other sections of medicine. And this morning I am using these three patients that we have had in our ward, to impress on your minds both sides of the question.

The first is an instance of gastric syphilis simulating carcinoma of the stomach.

G.T., *ætatis* sixty years, was admitted to Verco Ward on April 9, 1922. His history showed that three years previously, when working on a water scheme on the west coast, he had been suddenly seized with pain under the ribs which doubled him up for nearly two hours and was just as suddenly relieved by vomiting. For some months he had similar attacks about once a fortnight and then came to Adelaide. He had since worked as a labourer for a suburban municipality and occasionally had had similar attacks, but less severe. Three days before admission a severe attack had come on just after dinner and was most severe just under the ribs about 7.5 centimetres (three inches) to right of the mid-line. It lasted two hours and he then vomited undigested food and mucus and this relieved him. He rested next day and felt quite well, but on the next day after a light meal had a similar attack. Slight attacks recurred during the day and he was sent into hospital. His bowels were usually constipated; there had been no trouble with micturition. His past history showed that he had had small-pox, whooping cough, measles and influenza in childhood and "catarrhal jaundice" in 1915 which left him yellow for four months.

He was a smoker and a moderate drinker and denied any past venereal infection. His wife was healthy, but had had two miscarriages during the past twelve years; three daughters were healthy and one son had died of typhoid at the age of five months. He was not aware of any loss of weight.

Examination showed a sallow looking middle-aged man, with no marked wasting, asthenia or jaundice. His teeth were in bad condition—black coloured stumps with pyorrhœa—and his tongue was furred. Examination of heart and lungs revealed nothing abnormal; the abdomen showed epigastric pulsation, no distension, no tenderness or tumour, no enlargement of liver or spleen. There was an inguinal hernia on the left side for which he wore a truss. There was no abnormality in the nervous system. Below the right knee there was an extensive area of serpiginous ulceration which was evidently syphilitic in nature, although he had denied venereal disease. A test meal showed total acidity to be 43 and free hydrochloric acid 19; there was no occult blood. Radiographic examination after a bismuth meal showed no five hour residue in the stomach, the meal lying wholly in the caecum. On refilling there was shown a permanent filling defect at the pylorus, encroaching on the stomach mostly on the upper aspect; the interpretation was given as "pyloric carcinoma without stenosis." His blood gave the Wassermann reaction.

In spite of this very definite radiographic pronouncement, the question was raised in the light of the undoubted syphilitic ulceration of the leg, the Wassermann reaction of the blood and the normal acidity of the gastric contents, whether what was seen in the stomach could not be due to a gummatous ulceration, similar to that on the leg. Syphilitic ulceration of the stomach may be accompanied by a low or complete absence of acidity as in cancer, but in my experience a gastric carcinoma of so long standing as this history suggested would almost certainly produce achylia.

The patient was accordingly kept on the mixture of perchloride of mercury and potassium iodide which had been given to him for his skin condition, and no strict attempt was made to diet him. He remained free from pain for nearly three weeks, but a fortnight after admission his mixture was left off because of his severe headache which was thought possibly to be due to the iodide. Four days later he had an attack of pain lasting about an hour, but without vomiting and was put back on his mixture. Another attack came on a week later and next day injections of "Novarseno-billon" were begun and repeated twice weekly. At this time certain work was being done in the ward on blood sugar curves after food in gastric carcinoma and accordingly on May 16 he was given one hundred grammes of glucose at 9.30 a.m. his blood sugar being 0.15% just before food, 0.21% forty-five minutes later and 0.24% in two hours time. This delayed curve rather corresponded to that in gastric carcinoma and as he had two attacks of pain again two days later he was given another bismuth meal. The report showed that the condition had not progressed, but that if anything the stomach filled with rather less defect than a month earlier.

It was accordingly determined to persevere with the same line of treatment. He was sent to the night clinic for continuance of injections and transferred to out-patients' department. He attended there for some months with steady improvement in health. He has now been working steadily at his old occupation for over a year, has had no attacks of pain, says that he feels better than at any time for three years before treatment and presents himself today in quite good health. An attempt has been made to get him to come up for another radiographic examination to compare present findings with those of two years ago, but he is so well that he declines to leave his work to do this.

In this case you thus have a clear illustration of the occasional value of anti-syphilitic treatment in suspected carcinoma and a possible explanation of some of those cases which have been pronounced cancer of the stomach and subsequently get better.

The second case comes under quite a different category.

J.H., *ætatis* thirty-two years, is a blacksmith, who was admitted to Verco Ward on March 18 last. He was admitted because of attacks during the previous six months of pain in the upper part of the abdomen, accompanied by vomiting. The pain generally came on after meals; he would vomit about half an hour after and this would relieve the pain which was sometimes of a shooting character, sometimes a dull ache. He would have periods, up to three days' duration in which he would have no pain. For the previous six weeks the pain had been more severe and vomiting more pregnant. He had never vomited any blood; the bowels had been constipated, but there had been no tarry motions. Recumbent posture relieved the pain, so that there had been no interference with sleep. There had been no trouble with micturition. Past history had been uneventful; venereal infection was denied.

Examination showed a thin-faced man, with prematurely grey hair, a furred tongue and some carious teeth. Nothing abnormal was detected in chest or abdomen, except some slight general epigastric tenderness and some gurgling on pressure in the left epigastrium. The axillary and inguinal glands were slightly enlarged, but the chief point noticed was the complete absence of knee jerks.

This last fact, combined with the intermissions of pain and vomiting raised the question of the symptoms being due to gastric crises in *tabes dorsalis* rather than a peptic ulcer as had at first been thought. I have before this impressed on you the necessity of always bearing in mind the possibility of such intermittent attacks of pain and vomiting being due to gastric crises. Only a few months ago you saw a case of a woman sent to the surgical wards for operation for gall stone colic, which more thorough examination showed to be undoubted gastric crises with absence of knee jerks, Argyll Robertson pupil, Rombergism and ataxy. And I have mentioned to you other patients, known to

have been operated on for supposed gastric ulcer, where the symptoms were really due to tabetic crises. Years ago, I saw a patient whose condition closely resembled that of the present patient and in whom the only other sign present for some months after these attacks developed was the absence of knee jerks as in this case. That particular individual was operated on elsewhere some two years later for supposed gastric ulcer and a gumma of the liver was revealed. So that this is one aspect of syphilis that must always be borne in mind in the diagnosis of gastric disorders. In this particular case, however, the close relation of pain and vomiting to food and the absence of any affection of pupils or other tabetic manifestations made us pause for further investigation along both lines.

No abnormality of sensation or of gait could be elicited. Although the blood gave the Wassermann reaction, examination of the cerebro-spinal fluid after lumbar puncture showed no increase in lymphocytes and gave no Wassermann reaction such as would be expected in tabes. On the other hand, the result of the fractional test meal showed a curve both of total acidity and of free hydrochloric acid that was well above the normal range, and the first radiographic examination showed a small five-hour residue in the stomach, with a deformity at the pyloric end of the stomach and a doubtful ulcer on the lesser curvature. This examination when repeated a week later was reported to show a projection of opaque material from the lesser curvature high up which was tender and was interpreted as a penetrating chronic ulcer high up in the lesser curvature, with a questionable reflex pyloric spasm.

These investigations placed the fact beyond doubt that the symptoms were due to an ulcer of the stomach. The remaining question was whether this was an ordinary peptic ulcer unrelated to the syphilitic infection which was revealed by the serological examination, whether it was entirely due to the syphilitic infection or whether it was a peptic ulcer whose healing was delayed because of the syphilitic infection.

The patient was started straight away on bi-weekly injections of "Novarseno-billon," his diet was restricted to milk and carbo-hydrate food without any strict Sippy régime, as is usually practised in our treatment of peptic ulcer; nor were any of the usual antacid powders given between meals. After a fortnight he was given the usual mercury and iodide of potassium mixture regularly. After three weeks in bed with complete absence of pain he was allowed up daily and at the end of five weeks was sent home apparently well, with instructions to keep on same diet and mixture and come up weekly to the night clinic for his injections. It is now a fortnight since he left hospital and he reports himself quite well, able to do his work and putting on weight. It has not been possible yet to have another radiographic examination made, but I think there is no doubt from the result of treatment that, even though the origin of his ulcer was independent of his syphilis, its healing was delayed by that infection and that the rapid improvement without excessive restriction of diet was partly due to the anti-luetic measures adopted.

In the third case I am unfortunately unable to show you the patient, but the *post mortem* specimens are intensely interesting. They illustrate in a very interesting way the opposite point which I said I wished to emphasize.

In this case, J.L., a labourer, *ætatis* seventy years, was admitted to Verco Ward in September last on account of breathlessness and cough of some weeks' duration. He was then rather a florid-complexioned old man with orthopnœa and physical examination revealed a much enlarged heart with a to and fro murmur at its base and râles at the base of both lungs. He admitted to having contracted a urethral discharge some thirty years before and said he had a sore of the penis at the time. A diagnosis of syphilitic aortitis was made and was confirmed by the blood giving the Wassermann reaction and by the X-ray findings which showed cardiac enlargement, widening of the ascending aorta and several patches of opacity in the lung, questionably specific.

He was very ill for a few days, the notes showing that he developed Cheyne Stokes respiration on the second day after admission. With continued rest and the administration of tincture of digitalis and mercury and iodide mixture he steadily improved and after five weeks was sent home without symptoms.

Until his readmission on March 31 of this year, he had worked steadily, he told us, at light labouring work. Three weeks before he was re-admitted he was attacked with sudden constipation. He had no action of the bowels for four days and the medicine taken caused great pain. The attacks of abdominal pain had persisted up till admission, he had lost his appetite, had lost weight and strength, but had not vomited.

Examination showed marked loss of colour and of flesh as compared with six months previously. There was the same enlarged heart and to and fro murmur, though less forcible. In the abdomen there was now palpable a smooth transverse sausage-shaped mass, about twelve centimetres (four inches) in length, lying straight across the lower part of the epigastrium, as if in the transverse colon chiefly situated to the left of the mid-line. This mass moved with respiration and could be rolled between the fingers slightly from side to side and from above downwards, as though attached more deeply. It was not tender and its most marked characteristic to the touch was its rounded smoothness as opposed to the usual irregularity of a malignant growth. From the situation, the history of pain and constipation and his past history, it would have been quite easy to jump to the conclusion that here was a gumma invading the walls of the transverse colon and setting up a certain amount of obstruction. On the other hand, in that case there should have been evidence of hypertrophy or distension of the proximal bowel which was not present. His blood count showed his red cells were reduced to 1,900,000 with 21% hæmoglobin; though the cells were normal in shape and size. The fractional test meal showed a faint trace of blood in fasting juice. No free hydrochloric acid was found in any of the four specimens and lactic acid was present. Radiographic examination showed a large gastric residue after five hours, and on refilling a large filling defect of the pyloric antrum. On these findings the diagnosis of a gastric carcinoma with pyloric stenosis was made. The patient failed rapidly in strength during his stay in the ward and after three weeks died suddenly one morning.

The *post mortem* specimens which you see, demonstrate in a most vivid way the existence of the two conditions which were shown clinically in life. You see that the whole of the aorta is involved in a process which seems to be a mixture of the syphilitic process and the ordinary senile arterio-sclerosis. It begins at the end of the aorta, involving the aortic valves so that they are thickened and puckered, with consequent insufficiency. The heart is tremendously enlarged in consequence. The lining of the aorta shows the usual yellowish grey opaque swellings, but there are more calcified plates than one would expect in a pure syphilitic process. You will notice how the whole vessel wall is involved so that the vessel has lost its elasticity and seems like leather and in one part there is a general bulging like the beginning of an aneurysmal dilatation. The openings of the various branches stand out distinct without any obliteration and although the syphilitic process does not usually extend beyond the abdominal branches, you will

notice that this does and that even in the kidneys the cut vessels stand out prominently with the same leathery change. These changes are more characteristic of arterio-sclerosis than of a pure syphilitic aortitis.

Turning to the stomach you see that near the pylorus is a large fungating carcinomatous mass about twelve centimetres (four inches) in diameter attached to the posterior wall by a wide base. This is about 2.5 centimetres (one inch) in thickness from base to surface with soft edges which overhang the ulcerated surface. On closing the stomach you see how the impression of a round smooth sausage-shaped tumour was given in life by the smooth interior side of this mass being felt through stomach wall abdominal parietes. Behind the stomach you see a mass of enlarged aortic glands which extend upwards to the diaphragm and downwards to the bifurcation of the aorta. Section of these shows them to be firm and elastic and microscopical examination shows both these and the original growth to be carcinomatous in nature.

Here then too great an emphasis on the syphilitic factor might easily have led to an unduly favourable prognosis when the gastric symptoms arose. An interesting question is whether this carcinomatous growth was grafted on to an old gummatous ulcer. You know the opinions expressed by many that the majority of neoplasms in the stomach develope from an old ulcer and that this is especially true of syphilitic ulcers. That adds to the disappointment of not being able to get regular records of the first patient while free from symptoms. But discussion of that question would take us outside the point I wished to discuss this morning.

CONGENITAL HYPERTROPHIC STENOSIS OF THE PYLORUS AND PYLORO-SPASM.[1]

By P. L. HIPSLEY, M.D., Ch.M. (Sydney),
Honorary Surgeon, Royal Alexandra Hospital for Children, Sydney; Honorary Assistant Surgeon, Royal Hospital for Women, Paddington.

THE symptoms of pyloric stenosis are well known, the pathology of the condition has been accurately described, but the pathological physiology is not so clear. We do not yet know whether the symptoms are due to mechanical obstruction of the pylorus, caused by the tumour itself, or whether they are due to spasm of the hypertrophied muscle. Possibly both factors are concerned, but which is the greater remains in dispute. However the symptoms are due to the whole or portion of the stomach contents being prevented from passing the pyloric sphincter. This leads to dilatation of the stomach, increased peristalsis and as the food cannot pass on, it is forced through the cardiac sphincter and owing to the increased force of the peristalsis causes what has been termed projectile vomiting. As very little food enters the small bowel the motions first became thin, green and slimy (the so-called hunger stool) and later there is marked constipation. For the same reason little urine is passed and, as the infant wastes, the increased peristalsis of the dilated stomach is easily seen through the thin abdominal wall. Once having been seen, it cannot readily be mistaken for any other condition. In practically all cases the pyloric tumour can be felt if the examination be carefully made.

Diagnosis.

There are two conditions which are sometimes mistaken for pyloric stenosis, namely congenital atresia of the duodenum and pyloro-spasm.

Congenital Atresia of the Duodenum.

In congenital atresia of the duodenum the duodenum between the first and second portions and for about 1.25 centimetres (half an inch) or less of its length is represented by a fibrous cord in the centre of which is a canal. This will only admit a very fine probe and so there is a complete blockage to the onward passage of food. The first part of the duodenum in these cases becomes greatly dilated, the dilatation probably occurring before birth, a fact which at once distinguishes the condition from pyloric stenosis. The stomach, moreover, does not become hypertrophied and so vomiting is not projectile nor do we see the peristaltic waves, which are such a diagnostic feature of pyloric stenosis. The infants rarely live for more than a week and the few patients whose condition I attempted to relieve by gastro-enterostomy, did not recover.

Pyloro-spasm.

Besides the cases of definite pyloric stenosis, cases of severe and persistent vomiting are frequently encountered in young infants. This is apparently not due to overfeeding, nor can the quality of the food be blamed in any way. I have seen many of these cases in which atropine, gastric lavage, careful dieting *et cetera* have failed to stop the vomiting. The symptoms, however, differ from pyloric stenosis in several ways. The vomiting is not the forcible projectile type, constipation is not so marked and the infants pass plenty of urine. On examination for visible peristalsis, the peristaltic movements of the stomach can generally be seen on account of the thin wasted abdominal wall, but I have never seen anything that could possibly be mistaken for the peristalsis which is seen in pyloric stenosis. Of course no tumour can be felt in these patients. I have made exploratory incisions on four of these children, referred to me by physicians who had apparently come to the end of their resources and had failed to stop the vomiting. In none of these was there any thickening of the pylorus, but in three of them I divided the muscle, just as I do in pyloric stenosis. An exploratory incision only was made on the fourth. All these children recovered, but only after further prolonged medical treatment. Curiously enough three of these children were females and the sex of the fourth is not stated

[1] Read at a meeting of the Pædiatric Section of the New South Wales Branch of the British Medical Association on May 16, 1924.

in my notes. This is a point worth noting, when we consider the very high percentage of males in all recorded series of cases of pyloric stenosis.

Treatment.

The question of medical *versus* surgical treatment is still a debatable one. There are physicians who maintain, that if consideration is given to patients coming under treatment for the first time at all stages of the complaint, then medical treatment will give as good or even better results than surgical. No doubt there are cases eminently suitable for surgical treatment and others in which medical treatment offers the best hope. Dr. Leonard Findlay[2] has reported a series of patients treated medically with an 83% recovery rate and in the same article he mentions cures by other men, of 90% and another series of seventeen cases, with a 100% recovery rate. It would be difficult indeed for surgery to produce better results than these; but to get such results the infants would require to be under the care of skilled pædiatricians for a very long time. Where surgical treatment is successful, the infant is generally restored to normal health in a few weeks and this is a very big consideration in these cases. I am of the opinion that, if the symptoms have not lasted more than three weeks, surgical treatment offers by far the best chance, but after this it is quite possible that under suitable conditions medical treatment is justified for a time.

Operative Treatment.

Various operations have been designed to overcome the mechanical obstruction of the pylorus. The following procedures have been used for this purpose:

1. *Pylorodiosis.*—In pylorodiosis a portion of the anterior wall of the stomach is invaginated with the finger and then pushed through the pylorus.

2. *Loreta's Method.*—In Loreta's method the pylorus is forcibly dilated by an instrument introduced through a small incision in the anterior wall of the stomach near the pylorus.

3. *Pyloroplasty.*—In pyloroplasty a longitudinal incision is made through the pylorus and united so as to make a transverse scar.

4. *Nicoll's Operation.*—In Nicoll's operation the pylorus is forcibly dilated as in Loreta's operation. A V-shaped incision is then made through the muscular coats, but the mucous membrane of the pylorus is avoided and this incision is converted into a Y-shaped one and the sutures are inserted.

5. *Gastro-enterostomy* has been frequently used to obviate the obstruction.

6. *The Rammstedt Operation.*—The Rammstedt operation is most favoured at the present time for the treatment of pyloric stenosis, not that the results of other operations have not been as good in specially skilled hands.

G. F. Still[1] records a series of eighty patients operated on by Mr. F. F. Burghard with only six deaths, where forcible dilatation, similar to Loreta's method, was the method used. Such results are so striking that there is still room for discussion as to the best operation for these patients.

Personally I favour the Rammstedt operation. I always make the skin incision, about 0.8 centimetre (a quarter of an inch) to the right of the middle line in the epigastric region. The pylorus is delivered and an incision made through the thickened muscle until the whitish mucous membrane bulges slightly at the bottom of the incision. A portion of the tumour as free from blood vessels as possible should be chosen and it is best to begin the incision well away from the gastro-duodenal junction. Once the mucous membrane is exposed, the muscle right up to the gastro-duodenal junction can be divided, but as the transition from thickened muscle to normal duodenum is very abrupt, we have to proceed cautiously, so as not to open into the duodenum. The bleeding is very slight, if the correct site for the incision has been chosen. If, however, a bleeding point is giving trouble, it is easy to underrun the small vessel with fine catgut on a round needle, but it is better not

A SERIES OF NINETEEN CONSECUTIVE CASES OF PYLORIC STENOSIS.

No.	Name.	Sex.	Age on Admission.	Weight on Admission.[1]	Age at Onset.	Duration of Symptoms.	Operation.	Result.
1	R.L.	Male	25 days	2.5 (5½)	20 days	5 days	Rammstedt	Recovery
2	J.P.	Male	2 months	3.1 (7)	21 days	35 days	Rammstedt	Died 6 hours after operation
3	F.M.	Male	6 weeks	2.9 (6½)	14 days	28 days	Posterior gastro-enterostomy	Died 6 hours after operation
4	H.M.	Male	5 weeks	3.1 (7)	17 days	18 days	Rammstedt	Recovery
5	B.C.	Male	5 weeks	2.5 (5½)	12 days	23 days	Posterior gastro-enterostomy	Recovery
6	J.M.	Male	4 weeks	4 (9)	18 days	10 days	Rammstedt	Recovery
7	M.F.	Female	6 weeks	3.6 (8)	21 days	21 days	Rammstedt	Recovery
8	D.J.	Male	3 weeks	3.7 (8¼)	10 days	11 days	Rammstedt	Recovery
9	R.P.	Male	8 weeks	2.2 (5)	17 days	25 days	Rammstedt	Recovery
10	R.G.	Male	6 weeks	3.1 (7)	27 days	15 days	Rammstedt	Recovery
11	J.D.	Male	14 weeks	3.8 (8½)	35 days	63 days	Rammstedt	Recovery
12	H.S.	Male	6 weeks	3.1 (7)	14 days	28 days	Rammstedt	Recovery
13	H.M.	Male	14 weeks	4 (9)	21 days	77 days	Rammstedt	Recovery
14	N.B.	Male	5 weeks	4.1 (9¼)	21 days	14 days	Rammstedt	Recovery
15	L.O.	Male	9 weeks	2.9 (6½)	21 days	42 days	Rammstedt	Recovery
16	S.N.	Male	8 weeks	2.7 (6)	28 days	28 days	Rammstedt	Died 24 hours after operation
17	R.S.	Male	7 weeks	4.5 (10)	37 days	12 days	Rammstedt	Recovery
18	T.H.	Female	7 weeks	4 (9)	23 days	26 days	Rammstedt	Recovery
19	W.S.	Male	4½ weeks	2.5 (5½)	7 days	24 days	Rammstedt	Died 12 days after operation

[1] Figures given are kilograms; avoirdupois pounds given in parentheses.

to waste time in trying to stop all hæmorrhage, as it is essential not to take too long over the operation. The whole operation should not exceed ten to fifteen minutes. Before commencing the operation it is best to wash the stomach out with saline solution or a weak solution of sodium bicarbonate. Where the latter has been used, I have had trouble in delivering a dilated stomach, distended with gas, but the passage of a catheter into the stomach by the anæsthetist will simplify this procedure. The incision in the abdominal wall should be closed in layers. First the peritoneum is sutured, then three silkworm gut sutures are passed through skin, subcutaneous tissue and sheath of rectus, next the rectus sheath is sutured and after suturing the skin an anchored dressing is applied. The sutures can be removed after the tenth day. It is essential to keep the infant warm during the operation and warmth is perhaps the most essential part of the after treatment for the first few days. There is no objection in giving the breast after the effect of the anæsthetic has passed off, but it is better to limit the drink to about three minutes every couple of hours for the first twenty-four hours after the operation. Many of these infants vomit more or less for about twelve hours after the operation. If vomiting continues longer than this, it is generally stopped by washing out the stomach. Vomiting after this means, as a rule, that some muscle fibres have been left undivided or else it indicates faulty technique in preventing adhesions between small bowel and the incision.

The results after this operation have been eminently satisfactory and, provided the operation is not delayed more than two or three weeks after the first appearance of definite symptoms, the mortality should be very small indeed. When the operation is done after the symptoms have lasted more than four weeks, the mortality is much greater and in such cases I would recommend injections of saline solution or blood transfusion before operation. The results, of course, are better when the child has not been taken off the breast on account of the vomiting, as is so often done in these cases.

Summary of Cases.

There were seventeen males and two females. The earliest age at onset of vomiting was seven and the latest thirty-seven days. Fifteen commenced vomiting before the end of the third week and four after the fourth week.

There were four deaths after the Rammstedt operation. Two died six hours, one twenty-four hours and the other twelve days after the operation. In all these cases the symptoms had been present for more than three and a half weeks.

References.

(1) G. F. Still: "Congenital Hypertrophy of the Pylorus," *The British Medical Journal*, April 7, 1923, page 579.

(2) Leonard Findlay: "The Treatment of Congenital Hypertrophic Pyloric Stenosis, Medicine *versus* Surgery," *The British Journal of Children's Diseases*, January-March, 1923, page 1.

Reports of Cases.

ASPERGILLOSIS OF THE PLEURA WITH SCLEROTIUM FORMATION.[1]

By J. BURTON CLELAND, M.D.,
Professor of Pathology, University of Adelaide.

CASES of human infection by members of the mould genus *Aspergillus* are rare. The formation by the fungus of a dense compacted interwoven mass of hyphæ, constituting a sclerotium, within the human body must be much rarer, if not as far as recorded cases go unique. In the small amount of literature dealing with aspergillosis available to me I have not been able to find an example described. The small typical sclerotia in this case were extracted from the pleural cavity.

O. B., a private in the Australian Imperial Force, aged twenty-two years, was admitted to the Coast Hospital, Sydney, on May 18, 1918, suffering from an empyema following a gunshot wound of the right chest. On July 10, Sir Alexander MacCormick resected five centimetres of the eighth rib. Foreign material from the thoracic sac was submitted to the Microbiological Laboratory of the Department of Public Health for examination. On November 1, 1918, an X-ray report by Dr. W. B. Dight from No. Four Australian General Hospital showed a cavity in the chest approximately 7.5 centimetres long and 3.75 centimetres wide. On November 27, 1918, Sir Alexander MacCormick excised the sinus that remained, together with the adjacent portions of the seventh, eighth and ninth ribs and scraped the cavity. The cavity was about the size of a walnut and 6.25 centimetres from the surface of the body. Iodoform powder and gauze were inserted. The patient's temperature throughout was almost uniformly normal with occasional rises, but never above 37.7° C. (100° F.). The patient was discharged on February 19, 1919, and nothing further was heard of him. We are indebted to the Medical Superintendent of the Coast Hospital for kindly supplying the above information.

The material, removed from the empyema cavity on July 10 and submitted to the laboratory, consisted of a number of small firm brownish granules up to about 1.5 or two millimetres in diameter, with some greyish-brown discoloration of the gauze in which the material had been wrapped. Paraffin sections of the small bodies showed that they consisted of a dense felted mycelial network (constituting a sclerotium) with abundant fruiting bodies and spores on the periphery. The arrangement of the spores was that typical of an *Aspergillus, videlicet* conidiophore hyphæ terminating in ovoid formations supporting numerous claviform sterigmata. The conidia themselves had been dislodged from the sterigmata, but lay around these in numbers. The mycelial threads in the dense felted network forming the centre of the sclerotium were colourless, irregular and about 4.5 μ in thickness. Near the surface the mycelial threads became coloured brownish, were somewhat thicker and formed a layer from which the ovoid or elongated pyriform formations supporting the sterigmata arose. These "heads" had a diameter of 15 μ to 18 μ. Most of them were broken off and appeared to be subspherical to oval and about 15 μ to 17 μ long, but one perfect elongated pyriform head was found measuring 93 μ long, tapering towards the base into a gradually narrowed neck which accounted for 37 μ of this length. The sterigmata were closely set, claviform and about 5.5 μ long. The spores were brownish, smooth, spherical and about 3.5 μ in diameter. Castellani and Chalmers ("Manual of Tropical Medicine," Third Edition, 1919, page 1026) give descriptions of the various species of *Aspergillus* met with under pathological conditions. Our species agrees with *Aspergillus fumigatus* Fres., except that the conidia are distinctly coloured. The only other species it might be is *Aspergillus bronchialis* Blum., in which the mycelium is given as white, but in our plant

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 24, 1924.

the surface mycelial threads and conidiophores are brownish. Our specimens would thus seem to belong either to a new species or to one not yet known to be pathogenic to man.

On July 26, August 6, August 12 and November 28, 1918, further specimens of the discharge from the sinus were submitted in the hope that the *Aspergillus* could be grown. This was in every instance unsuccessful and no further evidence of the presence of the fungus was detected.

MEDIASTINAL AND RETRO-PERITONEAL LYMPHO-GRANULOMA (HODGKIN'S DISEASE).[1]

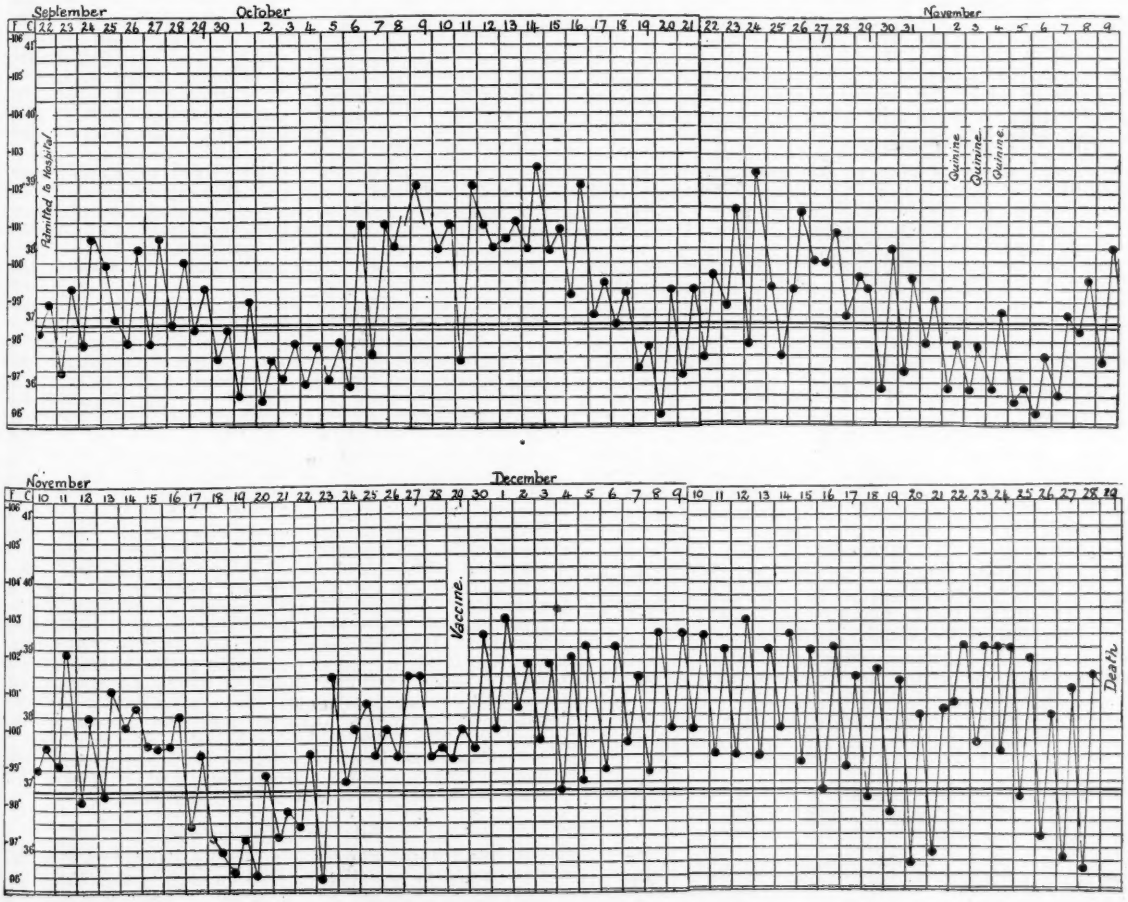
By A. GOODE, M.B., B.S. (Adelaide),
Peterborough, South Australia.

With a Pathological Report

By L. B. BULL, D.V.Sc.,
South Australian Government Laboratory, of Pathology and Bacteriology, Adelaide Hospital.

J.M., a male, *ætatis* twenty-two years, born in England, had suffered from the age of fourteen from nasal obstruction, bronchitis and modified asthmatic attacks. He reached Australia in 1918 and had a submucous resection done at the Adelaide Hospital in 1919, after which for a good while the chest condition was better. He had always been thin but active. He had never had hæmoptysis and there was no venereal history. He was shearing on a station near Broken Hill and about one month before his admission to the Peterborough Hospital on September 23, 1923, he had become rapidly ill and had had to take to his bed. The medical officer at White Cliffs gave a grave prognosis and said that his chest was "in a very bad state." On examination in the Peterborough Hospital he was found to be greatly emaciated, profoundly anæmic and slightly icteric. He had a frequent short cough and fairly abundant expectoration. His digestion was bad and the tongue coated, but the tonsils, gums and teeth showed no signs of disease. The glands in his neck were not enlarged. The chest was flattened at the apices of the lungs and rounded to barrel-shape at the base. Expansion was deficient, but the entry of air was free and there was good resonance. No adventitious sounds were heard except a few scattered and inconstant rhonchi and râles. There was evident emphysema. The sternal resonance was normal. The heart sounds were regular and pure but rapid. The abdomen was prominent. There was no ascites. The liver seemed soft and its edge could be felt 7.5 centimetres (three inches) below the ribs. The spleen was enlarged, but not greatly so and was firm but not hard. The glands in the axillæ and groins were not enlarged. The limbs were emaciated but otherwise normal. Bile and albumin were present in the urine which was alkaline, but there were no crystals, casts or blood cells. A fortnight later the albumin and bile had disappeared, the

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 24, 1924.



THE TEMPERATURE CHART OF DR. GOODE'S PATIENT.

icterus having become less. The jaundice was thus attributed to a gastro-duodenal catarrh causing obstruction. The blood showed a diminution of the red cells but no increase of the white cells. A blood film sent to the South Australian Government Laboratory showed variation in size of the red cells. The large mononuclear leucocytes were not increased and no malarial parasites were detected. The temperature chart showed a rough periodicity with fortnightly exacerbations. The patient had no rigors. He had copious sweats at times, but these were not of regular occurrence. The treatment was mostly palliative. Quinine was given in four 0.6 gramme (ten grain) doses daily during a remission in the temperature, but this did not prevent the temperature rising again. A vaccine made from mixed organisms in the sputum seemed to make the patient worse and so only one dose was given. He died on December 29, 1923.

The autopsy revealed no scarring or consolidation of the lungs. The liver was considerably enlarged and mottled with light and dark brown patches. The spleen was enlarged and felt firm and fibrous. From the posterior mediastinum to the lumbar region and closely adherent to the vertebral column was a chain of very greatly enlarged glands. These were encapsuled and the surrounding tissues were not invaded. The glands in the mesentery were not enlarged.

Pathological Report.

A mass of lymphatic glands from the mediastinum, a mass of lymphatic glands in the hilum of the kidney, the spleen and a small piece of lung were forwarded for histological examination. Some of the glands from the mediastinum were very large, the largest measuring ten centimetres across the larger and six centimetres across the smaller diameter. The mass of glands in the hilum of the kidney measured 11.5 centimetres long by four centimetres thick. The spleen weighed 530 grammes and was fourteen centimetres long by eight centimetres at the thickest portion.

Sections of the lymph glands showed hyperplasia of the endothelial and connective tissue cells with a diffuse infiltration of lymphocytes and polymorpho-nuclear cells. These polymorpho-nuclear cells did not give the staining reactions of eosinophile leucocytes which they might possibly have been; imperfect fixation possibly was the cause of the loss of characteristic staining reactions. Scattered throughout were cells with large nuclei; there were as many as six to eight nuclei in a single cell at times. The fibrous tissue had undergone a hyaline degeneration. There was periglandular infiltration of the granulomatous reaction. Some of the glands were almost completely sclerosed, the dense fibrous tissue showing a hyaline degeneration. In the softer glands lymphatic hyperplasia was more in evidence. Sections of the spleen showed the same histological picture as the lymph glands. Sections of the lung showed peri-bronchiolar accumulation of mononuclear and polymorpho-nuclear cells. Many of the alveoli were filled with the same types of cells. There was some bronchiectasis and slight fibrosis.

Comment.

As Hodgkin's disease is probably an infective process it is possible that the virus in this case gained entrance through the damaged mucosa of the bronchioles and first affected the glands in the mediastinum.

HYDATIDIFORM MOLE OF THE BROAD LIGAMENT.[1]

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AND

N. WIGG,
Clinical Clerk, Adelaide Hospital.

Clinical History.

E.M., a girl, *ætatis* nineteen years, was admitted to the Adelaide Hospital on March 18, 1923, complaining of pains in the joints, especially in the knees. These pains had commenced in the shoulders some days before admission and had involved the spine, hip-joint, knees and ankles in succession. She also complained of frontal headache and giddiness on walking of several weeks' duration. Her illness had commenced seven weeks previous to admission with an attack of cold shivers after a sea-bathe and she had been confined to bed for the greater part of the time up to her admission. She had been subject to attacks of rheumatism for ten years, having to go to bed for about a fortnight each winter with pains in her joints. Her tonsils and adenoids had been removed in 1920. The menstrual history given showed no abnormality and she said there was no intermenstrual discharge. Examination revealed a pale, anæmic-looking girl, but with good nutrition and development. There was some degree of photophobia. The pupils were dilated, but reacted to light and accommodation. The tongue was red and moist, the teeth in good order. The temperature was 36.6° C. (98° F.), the pulse rate seventy-eight and respirations twenty in the minute. No abnormality was detected in the heart or lungs and the abdominal viscera were apparently healthy. Her knees were slightly hot, but not acutely painful and not swollen. There was, however, some limitation of movement in both knee-joints. The breasts were fairly large and were tense, but there was no undue pigmentation. The urine gave a slight positive reaction to the test for ketones.

On March 20, 1923, the pains in her joints had gone, but there was tenderness on pressure in several muscle groups, especially in the intercostals and rhomboids on the right side. The following day even this had gone and for the following three weeks she remained in this condition, complaining only of slight headache, tiredness and lack of appetite. On April 8, 1923, however, her temperature which had been normal since shortly after her admission, started to rise. On April 12 she vomited several times and on April 13 her temperature reached 38.9° C. (102° F.).

She now showed very slight signs of a right-sided facial paresis, most marked in the tongue and forehead. She had photophobia again, but there was no return of the joint pains. On April 17 the temperature reached 40.5° C. (105° F.). A white blood count on April 18 gave 4,000 cells per cubic millimetre and microscopical and cultural examinations of the urine were without result. On April 19 a Babinski sign was elicited in the right foot; Kernig's sign was not elicited. On April 20 lumbar puncture was performed, with no result. For the following three weeks her condition remained the same; she had rigors every three or four days, with elevations of temperature to 39.4° C. (103° F.) or 40° C. (104° F.) for a few hours only and without subsequent sweating. Between these attacks she felt fairly well, but vomited occasionally. On May 5, 1923, she complained of pain in the left groin, shooting to the knee, and she now gave a history of having received a kick in the left groin five months previously and said that she had been losing a little blood between her periods since then. There was no abdominal tenderness and no tumour was palpable. On May 7 a white blood count gave 5,000 cells per cubic millimetre. On May 8, under general anæsthesia, a gynæcological examination was made by Dr. Verco who reported that the uterus was lying back in the sacral curve and that there was a mass in the left fornix, attached to the uterus and probably cystic. Removal was recommended. On her return to the ward the patient was still unconscious from her anæsthetic. She was pallid, her lips were cyanotic and the breathing was shallow. The pulse could not be felt. She was treated for collapse and improved slightly, but shortly afterwards her condition became suddenly worse and in spite of all measures taken she died a few minutes later.

Post Mortem Findings.

At the autopsy there was no undue pigmentation of the nipples and no secretion escaped on compressing the mammary gland. In the peritoneal cavity there was about a litre of blood, partly clotted, traceable to a small bleeding point on the posterior aspect of the left broad ligament between the ovary and the Fallopian tube. The left para-

[1] Read at a meeting of the South Australian Branch of the British Medical Association on April 24, 1924.

metrium and broad ligament was occupied and distended by a soft mass which on section showed shreddy tissue, hæmorrhagic areas and small vesicles such as are characteristic of hydatidiform mole.

The mass extended from the ovary to the lateral wall of the uterus, the outer part of which was invaded. The mole seemed to have originated in the Fallopian tube near its junction with the uterus. The distal portion of the tube was unaffected. On section of the uterus the cavity was empty save that in and near the left cornu were two soft small polypoid masses. A somewhat devious channel of a diameter to admit a slate pencil was found to connect the uterine cavity near these masses with a small mass about one centimetre in diameter embedded in the uterine wall. This also proved to be placental. The *cervix uteri* was soft and plugged with mucus. The ovaries were normal. The left lung was peppered with hæmorrhagic spots. In the right lung, the basal rim showed several small collapsed spots each possessing a firmer central zone suggestive of an infarct. One of these on section showed a small firm hæmorrhagic mass the size of a pepper-corn. In the middle and upper lobes were two or three pea-sized areas with small hæmorrhagic plugs and congested peripherally. There were no lesions in the heart. The spleen was uniformly enlarged, rather soft and of a peculiar hazy greyish-red colour. The kidneys were pale from loss of blood, but otherwise normal. The liver, stomach and intestines were normal. The brain showed some congestion, but no other macroscopical change.

Microscopically, the hydatidiform mole showed the usual myxomatous appearance of the villi which were capped with very pronounced syncytial and Langhans cell layers. In the wall of the uterus near the small mass referred to were villi with very marked syncytial masses and a few in-wandering large cells, sometimes with several nuclei. An infarct-like area in the lung showed some fairly dense fibrosis surrounding a mass of blood clot; in this blood-clot were two or three pale bodies, possibly degenerated villi. The kidneys showed congestion of the glomeruli and marked cloudy swelling. In the liver was a considerable increase of lymphocyte-like cells in Glisson's capsule. No emboli were detected in the brain. The breast did not appear to be physiologically active although a small amount of secretion was present in some of the ducts.

Commentary.

At first, in view of the history of joint pains *et cetera* a tentative diagnosis of acute rheumatism was made and the patient treated accordingly. Against this, however, was the absence of any signs of endocardial affection, in spite of a prolonged history of "rheumatism." Also the nature of the joint affection was not that of rheumatic fever. In a few days, when the nervous symptoms developed, the diagnosis of acute rheumatism was withdrawn and that of tuberculous meningitis considered. The results of lumbar puncture, however, did not uphold this, nor did the periods of quiescence between the rigors. An ordinary vaginal examination done in the ward at this stage did not reveal much beyond the retroversion of the uterus. This examination was done to try to detect any gross tubal condition if possible or evidence of appendical trouble. The explanation of the symptoms in view of the *post mortem* findings is not easy. Possibly a toxæmia was produced by absorption of autolytic products in the growth, somewhat similar to those produced in eclampsia, according to one of the theories of causation of that condition. Multiple small emboli from the growth may have been responsible for the joint symptoms, but it is more likely that these were due to the toxæmia. There was never any swelling, tenderness or marked inflammation of any joint and the pains in them were always rather vague and very fugitive. The nervous symptoms were quite possibly embolic in origin, although the headache and photophobia may also have been toxic. The history of continued menstruation was quite possibly inaccurate, occasional small hæmorrhages having probably been mistaken for the menses or the marital condition of the patient may have induced her to give the history she did.

The pregnancy seemed to have been located in the extreme end of the left cornu of the uterus and in the adjacent uterine end of the Fallopian tube. The remains of placental tissue in the uterine cavity and its wall did not macroscopically or microscopically show definite evidence of hydatidiform change, though there was invasion of the wall by placental tissue. This change was very pronounced in the placental tissue in the Fallopian tube and filling the broad ligament. Rupture of the thin covering layer of the broad ligament had given rise to a rapidly fatal intra-peritoneal hæmorrhage.

The following case of hydatidiform mole, reported by Dr. G. R. West, in THE MEDICAL JOURNAL OF AUSTRALIA for April 20, 1918, bears some resemblance to our case. The patient was a single girl, *ætatis* nineteen years, who was admitted to the Adelaide Hospital on December 7, 1917, with a history of three months' amenorrhœa. It was ascertained that an abortionist had passed instruments on November 2, November 29 and December 1 and that this had been followed by slight bleeding. On December 4 severe abdominal pain set in, accompanied by a blood-stained vaginal discharge. The temperature was 33.1° C. (*sic*), pulse one hundred and twenty and respirations twenty-eight. A large tender mass could be felt reaching nearly to the umbilicus and inclining to the right. Vaginal examination showed that the mass was continuous with the uterus. Under ether anæsthesia the cervix was dilated and a sound could be passed to the left for ten centimetres. The uterus was curetted, but no chorion was removed. The posterior fornix was then opened and a puncture into the mass was followed by profuse hæmorrhage. The colpotomy opening was therefore packed and the growth and uterus together with the right adnexa removed. The patient recovered, except for the presence of a vesico-vaginal fistula. The mass proved to be a tubal pregnancy which had developed into a hydatidiform mole. It had invaded the wall of the tube and of the *fundus uteri*. It was growing in the broad ligament and destruction of the wall of the tube had caused hæmorrhage, thus explaining the blood clots and free vesicles seen in the peritoneal cavity at operation. Microscopical examination of the growth revealed no evidence of chorion-epitheliomatous change.

Reviews.

DIABETES MELLITUS.

IT has been said that, "when one talks diabetes, one thinks Joslin" and all who have had to treat diabetes in recent years, owe much to Joslin's books on treatment of that disease. The new, third edition of the larger "Treatment of Diabetes"[1] has been awaited with interest, for it was expected that it would embody the experience of much new method. The expectation has not been in vain, for the book is full of new matter.

First and foremost is a full discussion of all the problems involved in the use of "Insulin" with the result of experiences of a year of treatment by that substance.

Joslin is naturally enthusiastic about "Insulin" which he describes as "a priceless gift to the severe diabetic, provided he be intelligent and faithful" and he especially emphasizes its great value in the treatment of children. "Of forty-eight children cared for in the year, forty-six remain alive and the two deaths were accidental."

Of the continued need for careful dieting with the standard fixed at "slight under-nutrition" or at least a "minimum maintenance diet" Joslin is very emphatic. He is totally averse to the luxury use of "Insulin," but insists on the use of the minimum amount of "Insulin" necessary to control the minimum diet adequate to the needs of the individual.

It cannot be said yet that standards of dietetics in diabetes are firmly settled. There are many diverse roads to the desired end, but Joslin's ideas are in the main

[1] "The Treatment of Diabetes Mellitus, with Observations Based upon Three Thousand Cases," by Elliott P. Joslin, M.D. (Harvard), M.A. (Yale); Third Edition, enlarged, revised and re-written; 1923. Philadelphia and New York; Lea and Febiger; Sydney: Angus and Robertson, Limited; Royal 8vo., pp. 797, with illustrations. Price: 42s. net.

sound and any medical practitioner using this book as his guide to treatment will find all needful methods described and should be able to rejoice with Joslin to see "mere ghosts of children start to grow, play and make a noise and see their mothers smile again" and possibly read in the paper that "his young colonel with the Victoria Cross, after ten years of faithful dieting has nearly won the local golf championship."

Apart from the chapters on "Insulin" there is an admirable account of much recent work on the pathology of the disease, on respiratory metabolism, the use of lævulose and the modern conception of acidosis.

Very interesting is the chapter on the Newburgh and Marsh's low carbo-hydrate, low protein, high fat diet which was proving so useful in treatment before the introduction of "Insulin" and is still used by Wilder and others in severe forms of diabetes as a starting point in the treatment by "Insulin." There are also excellent chapters on the diabetic laboratory and a full account of all the necessary tests, both of urine and blood, and there are most complete tables of food values with sample menus and many recipes for tasty dishes with their food value in carbo-hydrate, protein and fat.

One of the most interesting discussions in the book is on the subject of surgery and diabetes. Joslin is strongly of opinion that, if surgery is required in a diabetic, it should be performed just as if there were no diabetes and it is the physician's duty to prepare the patient and carry him safely through the crisis of the operation. Especial emphasis is laid on the importance of trying to store glycogen in the liver before operation and its replacement as early as possible after operation. The use of "Insulin" enables us to do this and we can cordially recommend both physician and surgeon who may be called to treat a diabetic patient by surgical means, to read Dr. Joslin's remarkable results, both in the pre-"Insulin" days and after.

It is impossible even to hint at all the good things contained in this volume. Suffice it to say that it is certainly the best clinical manual on its subject in the English language and should be in the hands of every medical practitioner who has to treat this all too common disease. The book is delightfully easy to read, both by reason by Joslin's literary style and the excellence of the paper and printing and we can only wonder at the amount of research and of detail that so busy a man has been able to place before his readers.

MEDICAL HISTORY OF THE WAR.

The volume of the official history of the war which is devoted to pathology, contains six hundred pages, two coloured plates and many illustrations.[1] It is more than a mere historical account of administration and events of interest in this branch of the Medical Services. Each of its twenty-three chapters is the work of an acknowledged expert and the book can be taken as the last word in the investigation of many problems of equal interest, whether regarded from the standpoint of military or civil practice. The contributions as a whole have an individual character since they deal largely with personal researches and investigations of their respective authors into the pathological problems of disease and of wound infection, and into the employment of sera and bacterial vaccines.

In the chapter on the physiology of wounds the work of Sir Almroth Wright and his collaborators is set out in detail and the ingenious devices used in these investigations are well illustrated.

Gas gangrene is dealt with by Adrian Stokes and the bacteriology of anaerobic infection by Miss Robertson. The histological changes in muscle at the site of gas gangrene are shown in a series of excellent micro-photographs. Amœbic dysentery is discussed at length by Lieutenant-Colonel D. Harvey in collaboration with Professor C. Dobell, while bacillary dysentery has been entrusted to Sir F. W. Andrewes. Colonal J. G. Adami has contributed the chapter on influenza, Colonel S. F. Cummins that on tuberculosis. An account of the experimental work on trench fever is contributed by Major McNee and on spirochætal jaundice by Major Stokes. War nephritis, cerebro-spinal fever, *encephalitis lethargica* and Vincent's angina are dealt with exhaustively and at the end of each chapter is appended an excellent bibliography.

The book is worthy of great praise and is of interest not only to the pathologist and bacteriologist, but to the physician and surgeon as well.

CLINICAL EXAMINATION.

IN the preface to his book "The Examination of Patients" Dr. Nellis B. Foster states that it was written in the belief that it would help practitioners of medicine. After reading the book we feel that the author is justified in that belief.[1]

Instead of the usual division into numbered chapters the book is arranged in sections which are not numbered. In these sections of which there is a large number, the author describes his methods of taking histories and examining patients. His methods are painstaking and thorough and his descriptions are clear and concise. Due stress is laid on the importance of studying carefully the patient as well as the history and results of the examination before resorting to laboratory and other aids to diagnosis. A critical attitude is adopted towards some of the modern methods of investigation.

The usefulness of the book is increased by the inclusion of numerous hints of practical value which are not usually found in books on physical examination. Descriptions of the examination of the blood and urine are omitted. Also in dealing with the heart and lungs the author does not give phonetic and other descriptions of the normal and abnormal physical signs nor does he give the theory of their mode of production. This book, therefore, cannot take the place of the books on physical signs with which students are familiar.

The omissions mentioned detract from the value of the work, but the author does what he sets out to do in a thorough and satisfactory manner and those who read his book, will find it interesting and helpful.

AN ENCYCLOPÆDIA OF MEDICINE.

THE tenth volume of the Encyclopædia Medica includes articles on terms from "obligate" to "potassium." In the present volume the standard set by the preceding numbers is sustained.[2] It contains a large number of carefully written special articles as well as the minor articles written by the editorial staff. Amongst the articles we find twenty-one pages given to affections of ocular muscles, thirteen to the œsophagus, twenty-one to diseases of the orbit, thirty-one to the palate, thirty to the pancreas, eighty-four to paralysis, forty-one to parasites, twenty-two to the pelvis, eight to pensions, fifteen to diseases of the pericardium, thirty-one to the peritoneum, twenty-three to the pharynx, thirty-five to plague, twenty-three to diseases of the pleura, fifty-two to pneumonia and twenty-seven to *post-mortem* methods. It seems rather anomalous, however, to find the small space of two pages given to palpitation and disordered action of the heart when we find as much as seven pages devoted to the surgery of the pituitary gland! But these anomalies are found in all encyclopædias and are almost inevitable. Plastic surgery is well described in this volume. It occupies nine pages and is illustrated by no less than six excellent plates.

[1] "History of the Great War Based on Official Documents: Medical Services—Pathology," Edited by Major-General Sir W. G. Macpherson, K.C.M.G., C.B., LL.D.; Major-General Sir W. B. Leishman, K.C.M.G., C.B., F.R.S., LL.D.; and Colonel S. L. Cummins, C.B., C.M.G., LL.D.; 1923. Edinburgh: 120, George Street; His Majesty's Stationery Office; Demy 8vo., pp. 600, with illustrations including two coloured plates. Price: post free: 21s. 9d. net.

[1] "The Examination of Patients," by Nellis B. Foster, M.D., 1923. Philadelphia and London: W. B. Saunders Company; Melbourne: James Little; Demy 8vo., pp. 253, illustrated.

[2] "Encyclopædia Medica," under the General Editorship of the late J. W. Ballantyne, M.D., C.M., F.R.C.P.E. (Volumes I.-VIII.) and Alexander Goodall, M.D., F.R.C.P.E. (Volumes IX., X.); Second Edition: Volume X., "Obligate" to "Potash"; 1923. Edinburgh: W. Green and Son, Limited; Sydney: Butterworth and Company (Australia), Limited; Royal 8vo., pp. 672.

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Secret Remedies.

PATENT medicines are not necessarily harmful or fraudulent. The medical profession, however, is wisely suspicious of these mixtures or compounds introduced for the pecuniary benefit of the manufacturers. Unfortunately medical practitioners have become large users of proprietary remedies and patent medicines. A prescription can usually be written containing pharmacopœial or non-officinal medicaments of known composition which will yield a better therapeutic action than any of the stock formulæ of the patent medicine proprietor. The art of prescribing seems to be almost a lost art. The result is that patients pay an exorbitant price for medicines without any commensurate gain in curative effect. It may be admitted that some proprietary preparations possess special virtues, either owing to a fortunate chemical process involved in the manufacture or owing to a happy combination of drugs which yields the desired pharmacological action. A medical practitioner is not justified in employing secret or proprietary remedies if he is ignorant of the ingredients or of their therapeutic value. It is by no means uncommon even for physicians of high standing to prescribe these remedies without any knowledge of their composition, merely because they have tried them and have acquired a belief in their efficacy. A fuller knowledge would at times disclose the fact that the therapeutic action depends not on pharmacological activity, but on the power of suggestion or that equally good results could be obtained for much less money from the use of a simple mixture containing the active drug of the proprietary preparation. It has been the policy of THE MEDICAL JOURNAL OF AUSTRALIA for several years to refuse advertisements of all proprietary medicines manufactured in Australia until a thorough investigation of the manufacture and a careful consideration of the ingredients by competent pharmacologists reveal the genuineness of the claims made on behalf of the preparation by the manufacturers. In regard to imported remedies, only those are advertised in our columns which have satisfied the Journal Committee of the British Medical Association in England as being worthy of a place in the advertisement pages of *The British Medical Journal*. Exception is taken at times even to preparations of which advertisements have appeared in *The British Medical Journal*, when information is available casting doubt concerning the reliability of the claims made for these preparations. Medical practitioners in Australia are thus given a guide concerning some of the reliable proprietary preparations. The appearance of the advertisement of those manufactured in Australia can be regarded as a guarantee of their efficacy, while reasonable reliance can be placed on the advertised remedies manufactured elsewhere. The journal is prepared to carry out investigations in regard to other proprietary remedies concerning which information is desired by medical practitioners, provided that the manufacturers are willing to submit the process to inspection. Under no circumstances can a remedy be advertised in the journal if the manufacturers are not prepared to disclose its composition.

Recently representatives of the proprietors and vendors of patent medicines urged the Chairman of the Health Commission of Victoria to endeavour to amend the regulations requiring that to every package containing a proprietary or patent medicine a label must be attached setting forth the ingredients for which therapeutic properties are claimed, the measure, number, volume or weight of each such ingredient contained in the dose recommended for an adult. This regulation, instead of being objectionable, is essential for the protection of the public. It does not go nearly far enough. In our opinion no patent or proprietary preparation should be offered for sale if the claims made on its behalf are untrue or exaggerated. Very few would stand the test. The daily press lends itself to a most undesirable traffic in useless, harmful or even fraudulent preparations. No attempt

is usually made to verify the claims boldly printed in the advertisements. These advertisements are a valuable source of revenue and no doubt it may be regarded as bad business to institute anything approaching a searching inquiry. Business morality seems to be elastic. The medical profession can do something to lessen the extent of the harm done by the traffic in secret remedies by refusing to prescribe or countenance the use of proprietary preparations of unknown composition or of worthless quality. The law must do the rest.

Current Comment.

BLACKWATER FEVER.

THE story of blackwater fever has fascinated the world of medicine for very many years, largely because of its mystery. Its association with malaria is admitted by all, but not so the significance of this coincidence. It has been established that in the great majority of instances it appears in persons who have been treated with quinine, but here again there are two opposing schools of thought. Those who deny that malaria is an ætiological factor, maintain that the accidental concomitance of the two conditions is sufficient explanation; quinine is exhibited to almost every person with malaria. Koch thought that quinine and not malaria gave rise to the hæmoglobinuria and renal involvement of blackwater fever oh the ground that quinine is a hæmolytic agent. The suggestion that blackwater fever and redwater fever of cattle are either the same disease or closely allied processes has little evidence in its support. Some observers have postulated an undiscovered protozoon as the causative organism, but until an independent virus can be demonstrated, such an hypothesis cannot command general acceptance. It is not surprising to find that in the presence of so much difference of opinion many more or less fantastic views have been elaborated to explain its ætiology. There are many facts suggestive of causal connexion between malaria and blackwater fever. In the first place the latter has not been observed in a person who has not been infected with malaria. Stephens admits that malarial parasites have been found in 95% of persons suffering from blackwater fever. Men working in West Africa maintain that it is impossible to exclude a malarial infection in anyone with blackwater fever. In the next place it is known that the products of the plasmodium of malaria are destroyers of erythrocytes. Jaundice and hæmoglobinæmia occur in uncomplicated malaria. In the third place the course of the fever is consistent with the assumption that to the usual effects of the malarial poison those referable to a massive destruction of red blood corpuscles have been added.

Dr. W. A. Young has recently offered an ingenious and by no means unreasonable explanation for the occurrence of blackwater fever.[1] He starts his hypothesis by accepting the physiological dictum that the life of a normal erythrocyte is about ten days. The death and disintegration of the cells and the removal of the liberated hæmoglobin takes place normally without the production of hæmoglobinuria. His next premiss is that red cell destruction in malaria depends on the action of the toxin or poisonous product of the plasmodium and of the plasmodium itself. The rate of destruction by toxin would be proportionate to the concentration of the toxin and would be intense in those situations where sporulation is taking place. *Plasmodium falciparum*, the tertian parasite of West Africa, gives rise to thirty-two merozoites. Allowing for the incidental failure of 75% of the merozoites, he produces some hypothetical figures to support his contention that the rate of destruction of the erythrocytes containing the parasites must be increasing in geometrical proportion. He calculates that one parasite would be capable of destroying at least two million erythrocytes in seven days. When every tenth cell is infected on the first day, the total number of cells in the body would be destroyed in thirty-five days. The action of toxin would be at least as rapid. Against this the rate of regeneration must be taken into account. But this takes place in arithmetical proportion and is in consequence slow. He therefore presumes that when the infection is severe, the condition spoken of as blackwater fever would follow within a relatively short time unless the patient dies before of cerebral malaria or hyperpyrexia. In other words blackwater fever must occur in patients with untreated severe malaria, provided they live long enough.

This hypothesis without further considerations would not account for the fact that Europeans are frequently attacked by blackwater fever, while the more immune natives are rarely affected. Dr. Young points out that the European usually has taken a relatively large dose of quinine when blackwater fever sets in. Quinine is a protoplasmic poison. Small doses do not produce visible change in healthy cells although they appear to lead to a condition spoken of as polychromasia. The plasmodium in severe infection has destroyed a large number of erythrocytes and has left many more in a damaged condition. The patient has been taking quinine irregularly; he then takes a large dose and the blood cell destruction is accelerated in consequence. The quinine attacks the plasmodium as well as the blood cell. The free merozoites find themselves in serum containing quinine and soon disappear. Dr. Young thinks that normal serum has some power of killing the parasites. It would thus seem that a considerable supply of malarial toxin with quinine added would be the chief determining factors of the attack. He attaches much importance to the erythrocyte in polychromasia,

[1] *The Journal of Tropical Medicine and Hygiene*, December 1, 1923.

the polychromatocyte. These considerations lead him to the conclusion which is supported by his clinical experience, that the optimum prophylactic dose of quinine must be that quantity which prevents a single malarial sporozoite from surviving in the blood stream. This varies with the intensity of the infection, the state of health of the individual and the temperature reaction. He expresses the opinion that if due regard be given to the presence of polychromatocytes in the blood and to the necessity of varying the prophylactic dose on scientific principles, blackwater fever or malarial hæmoglobinuria could be prevented altogether. His views demand attention, because they are entirely consistent with clinical experience and logical reasoning.

DRAINAGE IN ACUTE APPENDICITIS.

THE question of drainage in acute appendicitis is one which presents many difficulties to the inexperienced surgeon. Even those who have been engaged in surgical practice for some considerable time, are often unable to come to a prompt decision when faced with an acutely inflamed appendix and a quantity of turbid fluid lying free in the peritoneal cavity. Having arrived at a decision, many men would find it hard to explain the exact reasons for the adoption of a certain line of action. The matter is one about which it is well-nigh impossible to dogmatize for anyone. Yates in 1905 endeavoured to show that the peritoneal cavity could not be effectively drained and Andrew in 1912 reported success following closure of the peritoneal cavity in sixteen out of seventeen cases. Although he advocated abolition of the drainage tube as a routine measure in all cases of operation in acute appendicitis, he admitted that no hard and fast line of treatment was possible, but that each case should be treated on its own merits. He quoted Rutherford Morison in extenuation of early closing that the peritoneum has a resisting power not belonging to other tissues. Since that time the views of many surgeons have been modified and drainage is much more frequently discarded than was previously the case. The satisfactory results which may be achieved under certain conditions with closure of the abdomen, were brought home to many surgeons in the treatment of gun shot wounds of the abdomen during the Great War.

That there are many guiding principles of importance has lately been shown in the Hunterian Lecture delivered before the Royal College of Surgeons of England by Mr. R. St. Leger Brockman.[1] Mr. Brockman refers in the opening part of his paper to the dictum of Lawson Tait: "When in doubt, drain." He says that although such advice is admirable, doubt spells ignorance and that to act according to the ruling of such a motto is to spend a life of surgical stagnation. The presence of a purulent exudate has from long continued custom necessitated the use of a drainage tube. The sole advantage of such a practice has been described as the hypnotic influence of the presence of a tube on the surgeon himself. The surgeon often thinks that a fatal result occurring after closure of the abdomen might have been avoided if drainage had been used. Mr. Brockman regards this as untrue unless closure has taken place in the presence of an actual abscess. Death in such a case will be due to the absence of what is known as the resistance of the patient. Several advantages are gained by dispensing with the use of drainage. In the first place the occurrence of fæcal fistulæ is much more frequent when drainage tubes have been used. Residual abscess is more likely to occur and secondary hæmorrhage is seen more often after drainage. In one thousand cases of acute appendicitis reviewed by Mr. Brockman acute intestinal obstruction occurred in six instances in patients who had been discharged from hospital. Obstruction was caused by bands and in every instance drainage had been used at the primary operation. Mr. Brockman points out that the type of adhesion giving rise to this trouble is the outcome of chronic rather than acute infection. The drainage tube is soon cut off from the general peritoneal cavity by adhesions, a residual abscess is formed in which the organisms die, pus becomes inspissated and organized and the newly formed fibrous tissue becomes stretched and a band is formed.

Mr. Brockman explains the resisting power of the peritoneal cavity to infections as due to lack of tension. This results in an excess of constructive over the destructive elements of acute inflammation and explains the reason for the greater susceptibility of the upper portion of the abdomen to infection. The tabulated results of Mr. Brockman's analysis of one thousand cases is very interesting and shows that good results can be obtained from closure of the abdomen. He draws the deduction from his figures that children under the age of twelve will not stand closure as well as adults. This is because the attack for which the operation has been performed, is generally their first and they have not had the opportunity of developing a general or local immunity to such an infection.

Mr. Brockman refers to the work of Wilkie in regard to the immediate examination of the peritoneal exudate. He regards it of utmost value and says that absence of large mononuclear cells, their failing power for absorbing stains and the absence of phagocytosis are indicative of impending disaster. The greater the amount of fluid, the safer it is to close the abdomen, provided that the fluid is homogeneous in appearance. A gangrenous appendix with dry peritonitis, a blood-stained exudate and an abundance of flakes of coagulated lymph call for drainage. The epithelium of the peritoneum has sustained damage. An intact peritoneum must be left if the abdomen is to be closed. Mr. Brockman also discusses the condition of the appendix and caecum in regard to indications for drainage. He describes fully the method of drainage. He describes in detail the method of drainage of the abdominal wall in successive layers according to the method of Eisendrath and lays stress on its importance in the presence of an infection.

[1] *The British Journal of Surgery*, April, 1924.

British Medical Association News.

SCIENTIFIC.

A MEETING OF THE SOUTH AUSTRALIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Lister Hall, Hindmarsh Square, Adelaide, on April 24, 1924, DR. STEELE SCOTT, the VICE-PRESIDENT, in the chair.

Eosinophilia.

DR. THOROLD GRANT showed a female patient, aged twenty-eight years, who had been first seen early in February, 1924, when she had exhibited all the signs of a moderately severe secondary anæmia. Apart from pallor, the physical examination had revealed no abnormality. The blood count had been as follows: Hæmoglobin 37%, red cells 3,240,000 per cubic millimetre, white cells 58,000 per cubic millimetre. The differential leucocyte count had shown the neutrophile cells to constitute 23.5%, mononuclear cells 25.5% and eosinophile cells 51%.

Inquiry had elicited the fact that she had lived in Townsville, but that she had left the district three years previously. In childhood she had suffered from asthma, but there had been no attack for many years. Subsequent investigations had been made with the following results. The blood serum had not reacted to the Wassermann test. No reaction had occurred to the complete fixation test for hydatid disease or to the precipitin test. No ova nor worms had been found in the fæces on three examinations. The urine had contained no bilharzia ova. No filaria had been detected in the blood film taken at night.

A mixture containing iron and arsenic had been given. The red count and the hæmoglobin value had improved and the number of white cells had diminished, but the high percentage of eosinophile cells had persisted. No abnormal cells had at any time been seen in the blood film. On April 15, 1924, the hæmoglobin value had been 66%. The red blood cells had numbered 4,260,000 and the white cells were 22,500 per cubic millimetre. The eosinophile cells had then been 55% of the total white cells. Dr. Grant asked members for suggestions for further investigations.

Diathermy.

DR. G. H. BURNELL showed a patient suffering from recurrent squamous-celled carcinoma of the tongue, which he had treated by diathermy. The patient was a man of thirty-three years of age who had been first seen in June, 1923. His history showed that in May, 1916, he had had the right side of his tongue and the glands on the right side removed. A section of the tongue at that time had revealed a squamous-celled carcinoma. When seen by Dr. Burnell there had been a small hard ulcer on the right side of the tongue and another adherent to the angle of the jaw. The condition had not appeared to admit of treatment by ordinary means. The growth had therefore been destroyed by diathermy after ligature of the external carotid artery just above the superior thyreoid branch. Three weeks later a large flake of bone had come away from the mandible and the wound had then healed. At the time of the meeting there was a smooth soft scar in the region of the angle of the jaw.

Urethral Polypi.

Dr. Burnell's next case was illustrated by a coloured plate. The patient, aged forty-three years, married, had suffered for five years from very great frequency of micturition and strangury which had not been remedied by extensive gynæcological operations. Examination with the cysto-urethroscope had revealed five large polypi in the urethra and on the bladder neck. The ætiology of this type of growth had not been determined. The growths had been destroyed through the operating cystoscope by means of diathermy. Two months later there had been strangury and the patient was only voiding urine every three hours. She had gained 6.3 kilograms (fourteen pounds) in weight larely owing to longer periods of rest and sleep.

Dr. Burnell then explained shortly the principles on which diathermy depended and exhibited a portable diathermy machine for use with either direct or alternating current.

Hypernephroma of Kidney.

DR. BRONTE SMEATON read the clinical notes of a man, forty-one years of age, who had attended the Adelaide Hospital for treatment of a pain in the right knee. There had been tenderness, pain and disinclination to move the knee; semi-flexion was the position preferred. On examination signs of a tumour in the position of the right kidney and metastases had been found in the subcutaneous tissues, muscles and lungs. There had been no urinary symptoms and the cardinal symptom of hypernephroma, hæmaturia, had been absent. Dr. Smeaton said that in his experience the presence of hypernephroma, however small, seriously impaired the function of the kidney. He quoted a case in which a kidney containing a hypernephroma two and a half centimetres (one inch) in diameter had yielded urine with only 0.3% urea while the urine from the other side had yielded 2.8% urea. He said that in this case the urea of the affected side was 2.9% as again 2.5% from the sound kidney. The diagnosis had been made from the presence of the tumour, the cachexia and the metastases. A nodule had been removed from a muscle and the diagnosis had been confirmed by histological examination.

Dr. Smeaton said that an autopsy had subsequently been performed by Professor Cleland. Professor Cleland had found that there were a number of scattered nodules in the skin and subcutaneous tissues of the neck and of the arms as far as the elbows. A few similar nodules had been present in the pectoral muscles, in the muscles of the abdominal wall and in the subperitoneal tissues. The nodules on section had been white and somewhat cheesy. A roughly triangular tumour mass about thirteen centimetres by eleven centimetres in size had been found in the region of the right kidney. On section the inferior half of the kidney was found to be replaced by a soft tumour, yellowish in colour and showing hæmorrhagic patches. In the upper portion and surrounding a pyramid there had been present a few outlying whitish masses of growth. Below the renal pelvis and not communicating with it there had been a smooth-walled cavity filled with blood clot. Above the pelvis and opposite the upper pole of the kidney an almost diffluent hæmorrhagic and necrotic mass some four centimetres in diameter had been found. The renal vessels had not been involved. The lungs had presented a most unusual appearance. The surfaces had been studded with numerous, more or less spherical, projecting nodules which varied in size from a pin's head to about two and a half centimetres in diameter. The substance of both lungs had been œdematous and congested and many secondary deposits had been present corresponding in size and character with those on the pleural surfaces, but not so numerous. A small whitish nodule had been present in the posterior wall of the left ventricle. It had extended inwards from the surface almost to the endothelium. A secondary deposit the size of a pea had been present in the tissues near the head of the pancreas. The suprarenal glands, liver, gall bladder, stomach and intestines had not been involved.

In a histological report by Professor Cleland and Dr. L. B. Bull it had been stated that microscopically the histological picture was that of a Grawitz tumour. The cells itself varied considerably in morphology and in arrangement. The cells were sometimes elongated and almost spindle-shaped and with an alveolar arrangement. More frequently they were irregularly cuboidal to spheroidal and arranged in masses separated from each other by a delicate fibrous stroma, the cell masses occasionally showed a tendency to a papillary arrangement. Mitoses were very numerous. The cells had no particular close association with the vessels. Metastasis by the blood stream, which probably accounted for the deposits in the lungs and in the subcutaneous and intermuscular tissues, had probably occurred through erosion into pre-existing vessels rather than by ingress of cells into the vessels of the neoplasm itself. The cells did not suggest suprarenal cells in appearance; they much more closely resembled the cells of the renal tubules.

Preservation of Colour in Specimens.

PROFESSOR J. B. CLELAND showed museum specimens illustrating the preservation of colour by the coal gas-chloral hydrate method. This process had only been used in the laboratory for about two months. Most of the specimens treated showed so far complete retention of colour, though there was some tendency for the preserving fluid to become discoloured with hæmoglobin. It remained to be seen whether the colour would be permanently retained. The method of preservation was somewhat troublesome, but the cost was relatively very low. The following description of the method was based on an article by Oskar Klotz (*Journal of Laboratory and Clinical Medicine*, May, 1923—Extract from *Journal of the American Veterinary Medical Association*, September, 1923).

The specimens were placed in a special jar containing a fluid consisting of: Sodium chloride 8.5 parts, sodium bicarbonate 5 parts, "Formalin" 30 to 50 parts, water 1,000 parts. The jars used provide for an inlet tube extending nearly to the bottom of the fluid for the intake of the gas and an outlet at the top. After placing the specimens in the fluid and tightening down the cover the lower inlet was connected to the gas pipe. Glass covers luted with a thick rim of "Plasticene" had been employed for allowing the glass tubing for the inlet and for the outlet to enter the jar. This "Plasticene" soon deteriorated and properly constituted glass receptacles were preferable. Often the gas pressure was insufficient to force the gas up through the fluid so that it was necessary to attach a suction pump to the outlet. The flow of gas was continued for fifteen to twenty minutes, the process was repeated each day for from five to seven days. With specimens containing large quantities of blood the fluid became discoloured and should be renewed. The quantity of fluid should be about five times that of the specimens. It was stated that the use of carbon monoxide for the preservation of colour gave rise to a beautiful red hue which was quite stable and was not easily reduced by "Formalin." The blood cells did not lake and within a few days the blood was well fixed in the blood vessels, capillaries and tissues. Muscle hæmoglobin retained a good colour. When the specimen was well fixed, it should be immediately mounted in an air-tight jar containing the fluid described above, but to which had been added 2% of chloral hydrate. Professor Cleland said that he was adding a small quantity of potassium salts to aid in the retention of colour.

The specimens shown comprised: (i.) A slab of liver showing multiple secondary deposits from a carcinoma of the stomach in a male, *ætatis* forty-one years; (ii.) an enlarged spleen showing numerous large caseated tuberculomata from a girl, *ætatis* twenty-one years, who had in addition caseous tuberculous glands in the mediastinum and round the abdominal aorta, miliary tuberculous peritonitis and secondary miliary tuberculosis of the lungs.

Secondary Tuberculous Ulcers of the Rectum.

Professor Cleland then showed some specimens of the lower bowel. He said that tuberculous ulcers of the ileum and of the colon, secondary to advanced pulmonary tuberculosis, were common. It was not often, however, that the ulcers in the large bowel extended as far down as the rectum. In the present case they reached to within a short distance of the anal margin. For some time before death the patient had had diarrhœa with slimy yellow motions and three days before this event he had had a hæmorrhage from the rectum. As it was thought that a mass could be detected in this part of the bowel, a suspicion of the presence of a malignant growth was raised. The patient was a male, aged sixty years. He had been admitted to the Adelaide Hospital in July, 1923, and had then been ill for ten weeks. During his stay in hospital he showed typical symptoms of pulmonary tuberculosis and numerous tubercle bacilli had been present in the sputum. The patient had died on March 24, 1924. The autopsy had revealed advanced pulmonary tuberculosis, secondary tuberculous ulceration of the ileum, much ulceration and thickening at the ileo-caecal junction, scattered ulcers throughout the colon and in the rectum several typical tuberculous ulcers extending downwards to within 2.5 centimetres of the anus.

Hodgkin's Disease.

Professor Cleland then read the notes of a case of Hodgkin's disease in a patient who had been under the care of Dr. A. Goode, of Peterborough, South Australia (see page 635).

Hydatidiform Mole of the Broad Ligament.

Professor Cleland also read the notes of a case of hydatidiform mole of the broad ligament. The patient had been under the care of Dr. Angus Johnson and Dr. W. A. Verco (see page 636).

Aspergillosis of the Pleura.

Professor Cleland then read a short report on aspergillosis of the pleura with sclerotium formation (see page 634).

Gastric Syphilis.

DR. F. S. HONE then showed three patients with syphilis and gastric symptoms. He exhibited rediograms of bismuth meals in each case and read clinical notes.

Medical Societies.

MELBOURNE PÆDIATRIC SOCIETY.

THE EIGHTEENTH ANNUAL MEETING OF THE MELBOURNE PÆDIATRIC SOCIETY was held at Scott's Hall, Collins Street, Melbourne, on May 19, 1924, DR. R. M. DOWNES, C.M.G., the PRESIDENT, in the chair.

Annual Report.

DR. H. DOUGLAS STEPHENS, the Honorary Secretary, presented the Annual Report in which was reviewed a very successful year's work. Eight meetings had been held for the exhibition and discussion of interesting clinical conditions and pathological specimens.

One evening had been devoted to a consideration of basal metabolism. The introductory paper had been contributed by DR. NORMAN LORIMER and discussion was sustained by DR. C. H. KELLAWAY, DR. W. J. YOUNG, J. CHAMBERS and others.

DR. W. J. SAWYER, of the Rockefeller Institute of Medical Research, had given an interesting lecturette dealing with the hookworm survey and short papers had been read at other meetings by DR. R. M. DOWNES, C.M.G., DR. H. BOYD GRAHAM, DR. H. DOUGLAS STEPHENS and DR. REGINALD WEBSTER.

The success of the demonstrations arranged for the post-graduate class, visiting members of Congress and the Victorian Branch of the British Medical Association respectively, was recorded as due very largely to the capable organizing work of DR. J. W. GRIEVE and DR. H. BOYD GRAHAM.

The report concluded with an expression of thanks to the Committee of Management of the Children's Hospital, to the Matron and Nursing Staff and to all who had contributed to the success of the meetings.

Election of Office Bearers.

Office bearers for 1924 were elected as follows:

President: DR. WILLIAM DISMORE UPJOHN, O.B.E.
Honorary Treasurer: DR. ALAN B. MCCUTCHEON.
Honorary Auditor: DR. W. W. MCLAREN.
Honorary Secretary: DR. J. W. GRIEVES.
Hononary Assistant Secretary: DR. R. SOUTHBY.
Committee: DR. H. DOUGLAS STEPHENS, DR. R. M. DOWNES, C.M.G., and DR. H. BOYD GRAHAM.

Other Business.

In accordance with notice of motion given at the last Annual Meeting, DR. W. D. UPJOHN, O.B.E., moved:

> That the annual subscription be increased to one guinea for members of five years' graduation and remain at a half-guinea for those of under five years' graduation.

The motion was seconded by DR. MARK GARDNER and carried unanimously.

At the conclusion of the business members entertained at dinner DR. H. HUME TURNBULL and DR. M. D. SILBERBERG. Both these gentlemen had recently relinquished their positions on the honorary staff of the Children's Hospital.

The retirement of DR. H. DOUGLAS STEPHENS from the office of honorary secretary of the Society, which he had held since its inception, occasioned many highly appreciative references to the hard work and unfailing enthusiasm he had devoted to the interests of the Pædiatric Society for a period of eighteen years.

A MEETING OF THE ALFRED HOSPITAL CLINICAL SOCIETY was held at the Alfred Hospital, Melbourne on May 27, 1924, DR. J. P. MAJOR, the PRESIDENT, in the chair.

Actinomycosis of Back.

MR. A. J. TRINCA showed a man, aged twenty-seven years, who had complained of pain over the lower right ribs posteriorly since December, 1922. The onset of pain had followed chafing produced by carrying a sack over his shoulder which pressed against his back. Since April, 1923, when the streptothrix of actinomycosis had been detected, he had had almost continuous treatment with potassium iodide in doses up to eight grammes (two drachms) three times daily and two courses of autogenous vaccine treatment, the last of which he was receiving at the time of demonstration. Vaccine treatment had been limited owing to the difficulty in growing a sufficiently large supply of actinomycosis organisms. The disease had been only kept in check by the treatment. An X-ray film was shown.

DR. C. E. DENNIS suggested that as iodides and vaccines had not proved efficacious, radiological treatment should be tried. Recently radiologists had treated cases of actinomycosis with encouraging results.

Paralytic Dislocation of Hip Joint.

DR. HUGH TRUMBLE showed a boy, aged nine years, who at the age of one year and nine months had had infantile paralysis, mainly affecting the muscles of the right arm and left thigh. Recovery of muscle power had been good, but the left hip joint was dislocated. Movements were more extensive than normal in all directions at the hip joint. A skiagram showed the head of the femur well formed, the acetabulum somewhat less well. There was no evidence of any new joint formation.

The boy walked with a definite limp and appeared to experience more trouble in progression as he got older.

DR. F. K. NORRIS thought that an attempt should be made to replace the head in the acetabulum and suggested that the leg be extended by weights for a period of three months before replacement was tried.

Perthes's Disease.

DR. JOSEPH LOVE showed a man, aged twenty-two years, who had complained of pain in the right hip region for two years. Examination showed a firm swelling in Scarpa's triangle apparently due to some deep formation pushing forward the muscles. Movement was normal, but there was some pain at the extremes of movement in all directions. A skiagram showed that the head of the femur was flattened and mushroomed. Dr. Love thought that the case was one of Perthes's disease which had run its course without causing the patient much trouble.

DR. C. E. DENNIS quoted cases of Perthes's disease which he had seen in the army. There was always shortening of the femoral neck and flattening of the head. Often the condition gave rise to no trouble until an excessive strain was thrown on the joint, as in the patient before the meeting.

Encephalitis or Cerebral Tumour.

DR. W. S. LAURIE showed a boy, aged seven years, who in July, 1923, had a severe attack of measles. In September, 1923, he had begun to complain of headache, had vomited occasionally and had become shaky on his legs, bumping into furniture. He also seemed heedless and contrary. Then an alteration in his speech had been noticed and slight irregular movements in the limbs. There had not been any irritability or somnolence. He had been taken to the Children's Hospital where a diagnosis of tuberculous meningitis was made. At this time a partial reaction had occurred when his serum was subjected to the Wassermann test. No reaction had occurred with the von Pirquet test. The cerebro-spinal fluid had been normal and not under increased pressure. He had had paralysis of the external rectus of both eyes and slight facial paralysis. He had gradually grown worse, vomiting and headache had continued and had generally occurred first thing in the morning or after breakfast.

On examination the face had shown lack of expression but no facial paralysis, he had been able to whistle and use his facial muscles in an apparently normal way. There had been almost complete paralysis of the external rectus of both eyes. Movements of other eye muscles had been normal, but slight nystagmus had been present on upward movement. Pupils had been equal and had reacted normally. There had been no papillitis or optic atrophy. Palate and tongue movements had been apparently normal. Handgrips had been equal and good, but there had been some ataxia with fine actions such as writing and drawing. Abdominal reflexes could not be obtained. Knee jerks had been normal or slightly increased. The plantar reflex had been flexor in type. No ankle clonus had been present. The gait had been definitely ataxic. Slight ataxia in upper limbs had been accompanied by a tendency to overdo actions. Ataxia had not been increased by closing the eyes. Knowledge of position had not been impaired. No dysdiadokokinesia had been present. There had been well marked irregular choreiform movements, but without the typical hand grip or tongue protrusion of chorea. He had responded well to intelligence tests, but was slightly deaf. Ophthalmoscopic examination had revealed no optic neuritis or optic atrophy. On May 5, 1924, patient had been much worse. He had not been able to stand owing to ataxy and violence of muscular movements. Headache and vomiting had ceased. Muscular movements of the eyes were the same, but the pupils had tended to be dilated. Speech had been worse and ataxia and deafness were increased. Knee jerks had been greatly exaggerated. Ankle clonus had at times been present. Plantar reflexes had generally been extensor in type. The cerebro-spinal fluid had been under increased pressure, but otherwise had not been abnormal. A "good partial" reaction had occurred when the Wassermann test was applied to the blood, but no reaction had occurred in the cerebro-spinal fluid. No visceral disturbances had been present. Intelligence had still been very good. Ophthalmoscopic examination had shown pallor of the temporal half of the disc on each side. The nasal half had been normal—a descending type of optic atrophy.

DR. RINGLAND ANDERSON discussing the eye changes was of the opinion that the lesion was situated in the pons near the centre. The fact that there was bilateral paralysis of conjugate deviation supported this. On the other hand, the gait suggested the possibility of a lesion of the meninges possibly in the region of the *tentorium cerebelli*.

DR. M. D. SILBERBERG suggested the possibility of a basiliar meningitic condition, syphilitic in origin. This would explain the involvement of the sixth, seventh and eighth cranial nerves.

Obscure Nervous Condition.

DR. HENRY LAURIE showed a man, aged thirty-two years, whose illness had commenced four months previously with increasing weakness of the hands and arms. He had been in Caulfield Repatriation Hospital, where a diagnosis of peripheral neuritis was made. He had been transferred to the Alfred Hospital as his condition was not regarded as due to war service. His family history was good and his past history contained nothing of importance, except for a severe attack of trench fever in 1917. He denied excessive use of alcohol and there was no evidence of contact with lead or other substances likely to cause peripheral neuritis. On admission he was quite bedridden from weakness of his legs. There was great wasting of all the muscles of the upper limbs, especially of the intrinsic muscles of the hands, the thenar and hypothenar eminences on both sides being quite flat. There was

almost complete wrist drop on both sides. There was also considerable wasting of both lower limbs. No sensory changes could be ascertained with the exception of a doubtful deep tenderness on pressure on the calves. Deep reflexes were all present, but very difficult to obtain. Plantar reflex was not extensor in type. There was no fibrillation of muscles to be observed. Since admission about a month previously there had been slight improvement in the movements of the arms, probably due to the fact that for part of the time his wrists have been slightly extended by means of splints. Otherwise the condition had not changed. Dr. Laurie regarded the condition as one of progressive muscular atrophy in a stationary stage.

DR. A. V. M. ANDERSON pointed out that the tenderness and the improvement were against this diagnosis. He regarded the case as one of peripheral neuritis.

DR. M. D. SILBERBERG regarded the condition as a peripheral neuritis in view of the tenderness of the limbs and the presence of wrist and foot drop. Against the diagnosis of progressive muscular atrophy was also the absence of fibrillary tremors and also the absence in his opinion of atrophy of definite muscle groups.

Lenticular Degeneration Following on Encephalitis.

DR. W. S. NEWTON showed a woman, aged twenty-three years, who had been well until four years previously. At this time she had become ill with what was regarded as influenza, but diplopia had been present at the onset and ever since she had noticed loss of power in the right arm and leg. Some change in facial expression and speech had been noticed by relatives for the previous three years and this had progressed. Eighteen months previously she had noticed tremor in the right arm and leg, but this had since passed off. On examination the patient showed a mask-like expression and a slow drawling speech. The reflexes were normal, but the right superficial abdominal reflexes were not as brisk as those on the left. Definite rigidity of the right arm and leg and disturbance of automatic movements on right side were present. Dr. Newton regarded the case as one primarily of *encephalitis lethargica*, with lenticular degeneration as a sequel.

DR. A. V. M. ANDERSON agreed with this diagnosis and referred to similar cases in his experience.

DR. HENRY LAURIE remarked on the large number of patients suffering from *encephalitis lethargica* who often showed evidence some years later of various types of degeneration of the nervous system.

THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA.

A MEETING OF THE MEDICAL SCIENCES CLUB OF SOUTH AUSTRALIA was held at the University of Adelaide on May 2, 1924.

Simulium Larvæ.

PROFESSOR HARVEY JOHNSTON reported the discovery by Professor Cleland of simulium larvæ in swiftly running water near Hallett's Cove. He said that was the first occasion upon which the occurrence of simulium had been noted in South Australia. In some parts of the world these flies were a menace to stock industry and their bites might set up dermatitis in human beings. It was formerly suggested, but without any direct evidence, that these flies might act as carriers of pellagra. It had also been suggested again without evidence that nodule disease of cattle might be carried by these flies. In every instance the sole evidence consisted of the abundance of the flies in the localities where these diseases occurred.

PROFESSOR J. B. CLELAND stated that the occurrence of flies belonging to this genus had been reported in New South Wales, where he had found them feeding on wallabies. The larvæ had also been collected in New South Wales.

Kidney Lesion in a Fowl.

Professor Cleland reported that on *post mortem* examination of a fowl which had died of an illness of unknown origin, he had found numerous opaque granules in the kidney. Examination of sections showed that the Malpighian capsules were full of greenish-yellow acicular crystals. These crystals from their form and insolubility were inferred to be crystals of uric acid. It had been suggested that this might have been a *post mortem* effect; that the urine might have been especially concentrated and after death, as the body cooled, uric acid crystals might be precipitated from the urine *in situ*.

Professor Robertson had drawn attention to the recent work of Emmett and Peacock which showed that the fowl, unlike the pigeon, required a continuous supply of the fat soluble accessory foodstuffs and that if this was deficient in the diet, urates were deposited in various tissues, and particularly they reported, in the tubules of the kidney. This condition also occurred in the disease known as nutritional roup which was probably due to a lack of the fat soluble factor. The fowls were fed on household scraps, wheat and lucerne. It had been pointed out by Professor Robertson that household scraps might be deficient in the fat soluble factor and if the supply of lucerne chanced for a while to be deficient, the effects of deprival of this accessory might appear.

It was pointed out by Professor Cleland that the deposits in the specimens shown occurred in the glomeruli and not in the tubules of the kidney, thus differing to this extent from the results reported by Emmett and Peacock. This result was of further interest, because it appeared to demonstrate that uric acid was actually excreted in the glomeruli.

DR. L. B. BULL pointed out that the difference between the pigeon and the fowl in their requirement of fat soluble accessory might arise from the fact that the egg yolk was very rich in this accessory and that fowls laid many more eggs and more frequently than pigeons.

Aboriginal Remains.

DR. R. H. PULLEINE exhibited remains supposed to be those of a Tasmanian aboriginal found in 1920 in a cleft in the sandstone cliffs, forming the escarpment of the Mount Nicholas range, at Cornwall, near St. Mary's, Tasmania. Two dried hands with two humeri, two ulnæ and one radius had been found in a leather bag which was covered with sheets of stringy bark and weighted down by two stones. The remaining contents of the bag had been two pieces of skin, a quantity of what appeared to be dried viscera and two shells of *Pectunculus flabellatus*, a common bi-valve of the eastern Tasmanian littoral.

The remains had been found in a most sheltered position and exhibitor had recently visited and photographed the exact spot. He had been accompanied by the miner, Mr. Bradbury, who made the discovery. The spot had been found to be most sheltered and inaccessible to animals and absolutely dry.

Mr. Fuller had cut specimens of skin taken from the hands and found that there was such pigmentation as was found in a black race.

The importance of the discovery was its relation to the question whether the Tasmanians used the dried hands as objects of magic, as the Kurnal of south-eastern Australia used them (the so-called bretta or bret-bret). The presence of a long strip of skin removed from the arm with one of the hands to which it was still attached, favoured this view. The Tasmanians, moreover, were known to wear the lower jaw-bones, skull, other bones or parts of bones, as articles of ornament or magic, much as the Andaman islanders were reported to do at the present day.

Medico-Legal.

THOMPSON *versus* THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION.

IN our issue of April 5, 1924 (page 350) reference was made to the dismissal of the appeal by Dr. G. S. Thompson to the Privy Council against the verdict of the Full Court of New South Wales in favour of the New South Wales Branch of the British Medical Association. The judgement of the Lords of the Judicial Committee of the Privy

Council is printed in *The British Medical Journal* (Supplement, March 29, 1924, page 150). The importance of this judgement to the members of the British Medical Association and also to members of other voluntary societies is very great. Members should read it with care.

TRANSACTIONS OF CONGRESS.

THIS issue of THE MEDICAL JOURNAL OF AUSTRALIA does not contain a supplement. The publication of the Transactions of Congress will be continued in the issue of July 5, 1924.

INCOME TAX RETURNS.

WE have been requested by the Council of the New South Wales Branch of the British Medical Association to call the attention of members to the following matter. The Commissioner of Taxation of New South Wales advised the Council on May 6, 1924, that it had been decided to allow a deduction not exceeding £10 *per annum* in respect of the cost of medical journals and publications. The same deduction is allowed by the Federal Commissioner of Taxation (see THE MEDICAL JOURNAL OF AUSTRALIA, March 4, 1922, page 253).

Obituary.

JOSEPH CALLAGHAN.

WE regret to announce that Dr. Joseph Callaghan died at Lane Cove, New South Wales, on June 16, 1924.

Proceedings of the Australian Medical Boards.

TASMANIA.

THE undermentioned have been registered, under the provisions of *The Medical Act, 1918*, as duly qualified medical practitioners:

FRIEDMAN, HARRY, M.B., B.S., 1923 (Univ. Melbourne), Evandale.

HAMILTON, ALEXANDER ROBERT, M.B., Ch.M., 1923 (Univ. Sydney), Launceston.

PRYDE, DONALD, M.B., B.S., 1923 (Univ. Melbourne), Launceston.

WATERHOUSE, GEORGE ALAN, M.B., B.S., 1923 (Univ. Melbourne), Launceston.

WAITE, MARY ELIZABETH, M.B., B.S., 1924 (Univ. Melbourne), New Norfolk.

Books Received.

A MANUAL OF GYNECOLOGY AND PELVIC SURGERY FOR STUDENTS AND PRACTITIONERS, by Roland E. Skeel, M.D., A.M., M.S.; Second Edition; 1924. Philadelphia: P. Blakiston's Son and Company; Post 8vo., pp. 674, with 281 illustrations.

WHEELER'S HANDBOOK OF MEDICINE, by William R. Jack, B.Sc., M.D., F.R.F.P.S.G.; Seventh Edition; 1924. Edinburgh: Crown 8vo., pp. 644, with 34 illustrations. Price: 12s. 6d. net.

A DESCRIPTIVE ATLAS OF RADIOGRAPHS OF THE BONES AND JOINTS, by A. P. Bertwistle, M.B., Ch.B. (Leeds); 1924. Bristol: John Wright and Sons, Limited; Crown 4to, pp. 213, with 219 radiographs. Price: 17s. 6d. net.

A MANUAL OF PRACTICAL X-RAY WORK, by John Muir, B.Sc., M.B., Ch.B. (Pub. Health), in collaboration with Sir Archibald Reid, K.B.E., M.R.C.S., L.R.C.P., D.M.R.E. (Camb.), and F. J. Harlow, B.Sc., F. Inst. P., A.R.C.Sc.; 1924. London: William Heinemann (Medical Books), Limited; Royal 8vo., pp. 534, with numerous illustrations. Price: 31s. 6d. net.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, 429, Strand, London, W.C..

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney.	Australian Natives' Association. Ashfield and District Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham Dispensary. Manchester Unity Oddfellows' Medical Institute, Elizabeth Street, Sydney. Marrickville United Friendly Societies' Dispensary. North Sydney United Friendly Societies. People's Prudential Benefit Society. Phœnix Mutual Provident Society.
VICTORIA: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited Mutual National Provident Club. National Provident Association.
QUEENSLAND: Honorary Secretary, B. M. A. Building, Adelaide Street, Brisbane.	Brisbane United Friendly Society Institute. Stannary Hills Hospital.
SOUTH AUSTRALIA: Honorary Secretary, 12, North Terrace, Adelaide.	Contract Practice Appointments at Renmark. Contract Practice Appointments in South Australia.
WESTERN AUSTRALIA: Honorary Secretary, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Diary for the Month.

JULY 1.—New South Wales Branch, B.M.A.: Council (Quarterly).
JULY 2.—Victorian Branch, B.M.A.: Branch.
JULY 4.—Queensland Branch, B.M.A.: Branch.
JULY 8.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 9.—Tasmanian Branch, B.M.A.: Branch.
JULY 9.—Melbourne Pædiatric Society.
JULY 9.—Central Northern Medical Association, New South Wales.
JULY 10.—New South Wales Branch, B.M.A.: Clinical Meeting.
JULY 10.—Brisbane Hospital for Sick Children: Clinical Meeting.
JULY 11.—Queensland Branch, B.M.A.: Council.
JULY 11.—South Australian Branch, B.M.A.: Council.
JULY 15.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JULY 16.—Victorian Branch, B.M.A.: Council.
JULY 16.—Western Australian Branch, B.M.A.: Branch.
JULY 18.—Eastern Suburbs Medical Association, New South Wales.
JULY 22.—New South Wales Branch, B.M.A.: Medical Politics Committee: Organization and Science Committee.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, B.M.A. Building, 30-34, Elizabeth Street, Sydney. (Telephone: B. 4635.)

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